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STOKES ON FEVER . . . . . 16 PAGES.

LECTURE.

Lecture on Softening of the Brain. By J. HUGHLINGS JACKSON, M.D., Physician to the London Hospital and to the Hospital for the Paralyzed and Epileptic.

We very frequently hear the expression "softening of the brain." It is often used by educated patients; for many people who simply suffer slight and often but temporary nervous exhaustion think, always erroneously, that they have "softening," or are going to have it. It is really an expression of pathological application, but just as the symptomatic word "apoplexy" has come to have a pathological meaning (effusion of blood), so the pathological term "softening" has come—so, at least, it appears to me—to be used, even by some medical men, as a name for a certain rude clinical grouping of symptoms in cases in which there really is no softening. This use of the term is to be deprecated. Let me mention the symptoms of cases wrongly called "cases of softening." We see patients who have become excitable, irritable in temper, and desponding; they have found that their attention easily fails, and that they cannot do their accustomed work; they usually sleep badly; they have often what they call headache, but it is mostly not an ordinary headache, either in kind or in position; it is a feeling of pressure, or sometimes of burning, and its seat is the vertex or the back of the head; there is very often, indeed, a disagreeable feeling at the occiput and in the upper parts

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of the spine, more distressing than pain —an intolerable physical feeling; the queer feeling in the spine is often intermittent, and frequently comes on slowly with great depression of spirits. Altogether there is a strange mixture of "mental" and "physical" symptoms. Recognizing the group of symptoms I have mentioned as a fair clinical entity deserving particularly careful study, I do not see the evidence for the diagnosis that softening of the brain is the pathological change causing them. Such cases are called by the laity nervous debility, and often by medical men hypochondriasis. The symptoms, I think, indicate nervous exhaustion, beginning often in the sympathetic nervous system, and secondarily affecting the nutrition of the highest centres in the brain. Of course this is only hypothetical, for there is no morbid anatomy of such cases. Every one's conclusion as to their pathology must, therefore, be hypothetical. These symptoms are often produced by excesses, and especially by sexual excesses, and by "fast life" generally; they are sometimes suddenly developed by fright, and may be brought on by misery or overwork, either of the mind or the body, especially when the work is done under responsibility. Of course they occur most often in persons who inherit a weak temperament, who bear trouble badly, who are easily excited and easily depressed. In some of the cases the patients get quite well by simple common-sense care, and the delusion that they have softening vanishes. In the graver, prolonged, and ingravescent cases, I should think there was no softening of the brain, but rather greater firmness of it; atrophy of nerve-cells and fibres, with increase of connective tissue; there is some atrophy of the brain. In saying this I am not making a very strong statement. We often see considerable atrophy of the brain at post-mortem examinations on those who have died of non-cerebral disease, and whose mental condition has attracted no attention. Atrophy of brain is normal in old people; it is often seen in middle-aged drunkards and even in comparatively young people who have been long bedridden by wasting diseases not primarily involving the nervous centres. I mention this, as you may think the statement that there is some atrophy of the brain an extravagant conclusion as to the state of things in a man whose symptoms are those of prolonged and severe hypochondriasis or nervous debility, or whatever the right name or label is.

Be sure there is no softening in these cases. Indeed, I do not see how the diagnosis that there is actual softening of the brain is in any case to be possibly arrived at, *unless the patient has certain local paralytic symptoms, as hemiplegia, or some other symptoms implying a local cerebral lesion, such as affection of speech;* or, again, unless there be signs of cerebral tumour (severe headache, urgent vomiting, and double optic neuritis), or evidence of injury to the head. For, so far as I know, cerebral softening is always local; I know nothing of general or universal softening of the brain. To be warranted in diagnosing softening, you must have symptoms which point to local disease. I do not say that local cerebral softening cannot exist without localizing symptoms. I only say that in their absence you are not warranted in diagnosing its existence. We know that large parts of the brain may be destroyed without any marked local symptoms resulting; these parts may be destroyed by the process of softening without causing marked local symptoms. But in these cases the softening is mostly about tumours or other kinds of adventitious products. It is, however, almost an abuse of language to speak of these as cases of softening. The softening is, I suppose, a result of encephalitis about the tumour. We shall not refer to these cases again. For all practical purposes they belong to a distinct category. The cases which deserve to be called cases of softening are cases in which there is blocking up of cerebral arteries, or, which is infinitely rarer, of cerebral veins. Of these only shall I speak.

It is from the distribution of arteries, it is "for arterial reasons" so to speak, that there are localizing symptoms in

softening. The highest part of the motor tract (corpus striatum) and adjacent convolutions are usually the parts of the brain damaged or most damaged in softening, or simply the parts of the brain which often undergo softening. And this is because the artery which supplies these parts is an artery—the middle cerebral—which often gets blocked up, for reasons we shall afterwards state. Hence hemiplegia and affections of speech are the symptoms to be most trusted in the diagnosis of cerebral softening, for they are the symptoms producible by disease of the parts mentioned as being supplied by an artery which often gets blocked up. Curiously, however, unless there be valvular disease of the heart to, as it were, insist on softening by the process of embolism, cerebral softening is frequently not diagnosed in cases of hemiplegia and affection of speech when it often exists. Suppose that perfect hemiplegia comes on in a quarter of an hour without loss of consciousness in a patient past middle age, who has not renal disease, we are practically certain of local cerebral softening. There is in these cases softening by thrombosis—a commoner cause of local softening than embolism. Thus we see that the word "softening" is used by some for cases of nervous exhaustion and cerebral atrophy where there is not that pathological change, and, strangely, those who so use it very often do not diagnose softening in the really simple cases of hemiplegia coming on without loss of consciousness where it does exist. Sometimes, indeed, when the diagnosis is made that hemiplegia has been caused by embolism, because the patient is young and has valvular disease of the heart; there seems to be no clear notion in the mind of the diagnoser that he has before him a case of softening. There are two reasons for these mistakes. First, hemiplegia is too often attributed as a matter of course to cerebral hemorrhage, and softening is not thought of. This is, I think, because clot is oftener seen at post-mortem examinations in cases of hemiplegia, as it is a more speedily fatal lesion. We have more autopsies on patients who die from cerebral hemorrhage, because they die sooner, and therefore under our care. Nevertheless, hemiplegia *coming on without loss of consciousness*, is mostly caused by softening. I except cases of chronic renal disease, for hemiplegia in these cases, as a matter of fact, nearly always depends on effusion of blood, whether there be loss of consciousness at the onset of the paralysis or not. A second reason for the mistake in diagnosis is that there is a widespread impression that general mental symptoms *must* exist if there be "softening." I shall devote a great part of this lecture to show that this is an error, or at any rate, it is an error to suppose that softening of the brain *necessarily* causes *general* mental symptoms *directly*. Observe, I say directly, because I shall show that softening may be preceded by general mental symptoms, and may be followed by general mental symptoms. But in neither case do these symptoms depend directly on that softening. Observe, too, that I speak of *general* mental symptoms. I admit that softening directly causes special mental symptoms—loss of speech, for example. I deny that it directly causes general mental symptoms, although I admit, as I have said, that it may be preceded and followed by them. It is practically convenient to speak first of *special* mental symptoms *directly* resulting from softening; then of general mental symptoms following it. Although out of chronological order, it is more methodical to consider the mental degradations which may precede an attack of softening after speaking further of arterial changes.

Certainly there is not necessarily any direct connection betwixt cerebral softening and *general* mental symptoms. There need be no such symptoms at the onset. The typical case is that of the middle-aged man who, having gone to bed feeling well bodily and mentally, finds out when he gets up in the morning that he cannot use his left side, but who is as clear in mind as usual; yet that man has local softening, or rather thrombosis of an atheromatous artery supplying a part of the motor tract, which will therefore speedily soften. We are obliged to make some kind of arbi-

trary limit. We speak of cases commonly met with. But, as I shall tell you later, there may be deep coma from blocking of the trunk of the middle cerebral artery—coma as deep as that producible by large cerebral hemorrhage. A patient may have, however, special mental symptoms, without any insensibility, at the onset of softening, and as a direct result of it. Such cases I now consider.

Special mental symptoms directly resulting from softening.—The parts of the brain which most frequently soften are, as I have just said, the highest divisions of the motor tract; most often the corpus striatum and adjacent convolutions—that is, in other words, the parts supplied by the middle cerebral artery. If a large part supplied by the right middle cerebral artery be softened, there need, after the shock of the onset, be no obvious mental symptom of any kind. If a branch of the *left* middle cerebral artery be blocked there is a mental symptom, but then it is not a general one, but an exceedingly special one—viz., affection of speech (aphasia). Affection of speech *is* a mental symptom. A person who has lost speech has lost a part of his mind. Indeed, if the *trunk* of the middle cerebral artery be blocked there is very extensive softening, and a state very like imbecility as well as loss of speech; the patient's power of expression in all ways, and even the exhibition of states of feeling by smiling, etc., may be gone. Even then the symptom is a special one—loss of language or loss of speech.[1]

[1] I must insist that loss of speech is a mental symptom. For we speak not only to tell other people what we think, but to tell ourselves what we think; rather a proposition is the ending of a mental operation, and often the beginning of another. The aphasic can still think in some fashion; he remains able to do so, I consider, because, although speechless, he is not wordless; he has remaining the automatic and unconscious use of words in the right undamaged cerebral hemisphere. I believe, too, that there is a similar duality of Perception, or, rather, that the process which ends in perception is double. This process is not affected in cases of aphasia. There is, I think, chiefly from disease of the right posterior lobe, a mental state which I call "Imperception," corresponding to Aphasia in the Word series. I do not discuss this symptom in the text, for, besides other and better reasons, the topics necessarily to be considered are numerous.

General mental symptoms may follow local softening. We will for convenience speak separately of those following in a few days (these are usually temporary), and then of those following weeks or months after; these are often permanent. Remember the simple case we took for the purpose of illustration. A patient, about fifty, has become hemiplegic without loss of consciousness. This is the symptomatic statement; the pathological statement is that he has local cerebral softening.

General mental symptoms following a few hours or a few days after local softening.—After a short time the hemiplegic patient may begin to "wander," although at first, and usually all through his illness, he can pull himself together, clearing his mind of his fancies, and can reply to ordinary questions correctly. The mental symptoms are of a general character. There is not a loss of one faculty in particular, but a reduction of the whole mind to a more automatic condition. The patient "wanders" about his business, about the persons or places to which he is most accustomed (which are most automatic to him). He imagines that he is doing his work, and he may take strangers for those persons to whom he is most accustomed. I repeat there is not here loss of any one faculty, there is no special mental symptom. The patient is reduced to a more automatic condition of mind. There is Dissolution, using this term as the opposite of Evolution. Then, of course, much depends on the kind of brain the patient had before his illness. Old drunkards will have more absurd delusions, illusions, and hallucinations than persons who have not abused their nervous systems. My own belief is that this state results simply because the hemiplegic patient's brain power is slightly reduced in a universally lowered bodily condition, and that it is not caused by the very local disease of his brain. A local softening, practically an absence of brain (circumscribed) destruction, could not possibly cause such *active* mental symptoms as we have mentioned. These symptoms are, in my

opinion, often owing to debility. Let me explain how I think *debility* produces *active* symptoms. Explanation is required, as the expression seems paradoxical, and the expression "debility" wants definition. Probably the hemiplegic patient has, for theoretical reasons, been put on a spare diet, a diet next to nothing, although the very local damage in his brain has not seriously affected his digestive organs—not obviously affected them at all. The palsy has alarmed and depressed him, and he has not in consequence slept, or has not slept well. Very likely he has had a strong purgative, and perhaps blisters to the back of his neck. These mental symptoms are often due in great part to want of food. Very likely the poor fellow may have every day for years eaten and drunk far too much. Some people eat little during the day, and then "stun" themselves at dinner. If such a man be taken hemiplegic just before his dinner hour he would feel the want of food in the night. We should be cautious how we begin to change a man's habits, even bad habits, very soon after an attack of hemiplegia. If we do not keep up his arterial tension the parts of his brain *the furthest from his heart* will not get plenty of blood. The parts geographically furthest from the heart are the most intellectual; he may therefore become delirious if his arterial tension be much lowered. When I have spoken more about arterial changes, you will see that the brain of a man who suffers softening is often one of which the apparatus for nutritive supply has long been getting slowly defective. He is usually past middle age. There is arterial degeneration, and thus the circulation of distant parts will fail very easily. So far, we have concluded that there is defective blood-supply, and corresponding defect of action of the highest, that is of the most intellectual nervous arrangements. But obviously this negative condition could not account for *active* mental symptoms. Before I speak of the manner in which I believe the active symptoms to result, let me widen the field of observation.

I would earnestly beg you to bear in mind that I declare that such mental symptoms are not characteristic of cerebral softening; they are just as likely to occur with hemiplegia from clot as with hemiplegia from softening; they cannot be due directly to either; they cannot, as I have said, be due to circumscribed "destroying lesion" of any kind. But we can go far beyond this statement—we can say that these symptoms are rarely dependent on *primary* disease of the brain of any kind. In general physicians' practice very active mental symptoms (delirium) are rarely even associated with primary disease of the brain of any sort. Of necessity they imply something wrong in the brain, but the brain is suffering secondarily, "functionally," as the popular and inexact expression is. Such symptoms are common in the medical wards of a hospital. The patients in whom they occur are those whose primary ailment is not a cerebral one; they occur in phthisis, in chronic Bright's disease, in pneumonia, in erysipelas, and in pyæmia—in some cases of these diseases, I mean. As a rule, the more acute the disease, and therefore the more rapidly the brain suffers secondarily, the more active, the more uproarious and constant, the mental symptoms. They occur without a very high temperature oftener than with one. The temperature is often normal. Now we come to the explanation.

Such mental symptoms are on an utterly different platform from all other kinds of nervous symptoms. Remark, they are positive, or, as I said, active symptoms. Negative mental symptoms, such as loss of speech, imbecility, defect or loss of consciousness, are often owing *directly* to disease of the brain, but the most extravagant and most changing positive mental symptoms must in all cases, even where there *is* disease of the brain, be due to the action of parts of the brain which are *not* the seat of a lesion which produces *loss* of function. They are due to the action of parts which, except for over-excitement, are healthy. I am fully convinced that in all cases, from the insanity of slightest delirium to insanity ordinarily so-called, there is, as Dr. Monro says (in accordance with a principle many

years ago formulated by Laycock), a negative and a positive condition. There is loss or defect in the highest faculties along with exaltation of those lower and more automatic. It is even so in health; automatic mental action goes on during sleep or reverie. But we shall keep to actually morbid conditions. I wish to impress on you that it is the negative element only which is the *direct* result of disease. Now mark, the delirious patient is symptomatically in a double condition. The negative element is defect or loss of consciousness. Unfortunately such an expression often rouses thoughts about coma; but be sure that there are all degrees, from the slightest and most transient confusion of mind (defect of consciousness) to deepest coma. Now, the defect of consciousness is the condition produced directly by loss of arterial tension leading to failure of the highest centres—those most distant from the centre of the circulation. With regard to the causation of the positive symptoms (the delirious ravings, etc.), we may apply the principle Anstie stated when he said "that the apparent exaltation of faculties is due to removal of controlling influences," or, as Thompson Dickson said of delirium and mania, that "they result from loss of control," or as Rutherford says,[1] "to diminution of inhibitory power." When a man is delirious his negative state is, I repeat, that his highest nervous arrangements in the cerebrum, the sub-strata of consciousness, are more or less put out of use; he has defect of consciousness. In any general lowering of health the very highest nervous arrangements are the first to fail. (Principle of Dissolution.) The positive or active symptoms, the illusions, delusions, and grotesque actions, are owing to the action of the lower nervous arrangements, which, except for over-excitement *permitted* by the loss of control, are healthy. (Principle of Loss of Control.)

You may reply that the active mental symptoms may be attributed to active disease about the original lesion—about the softening or the clot. This is contrary to the evidence. It is a grave error in any case to put these symptoms down as a matter of course to meningitis, encephalitis, or to any kind of acute primary head affection. It is a deplorable error if it leads to severe purgation, to blistering the back of the neck, leeches, and low diet. The characteristic symptoms of encephalitis, meningitis, and acute brain disease generally are not mental, but physical. Thus, besides gross motor affections, convulsion, and paralysis, there are alterations of circulation, respiration, temperature, and constipation, vomiting, etc. Do not forget this seemingly paradoxical statement, that the trustworthy symptoms in the diagnosis of acute and primary disease of the organ of mind are physical, and that the untrustworthy symptoms for that diagnosis are mental.

General mental symptoms slowly developing weeks or months after an attack of softening.—These certainly follow at too great an interval to be erroneously attributed to the local damage which caused the hemiplegia. They often develop when the hemiplegia has become chronic. A general mental and bodily failure often follows a severe attack of hemiplegia, the result of local softening. It is not due to that softening nor to any extension of it; it is, I suppose, a result of secondary atrophy of the whole or of a large part of the hemisphere in which the local softening lies. But remark that widespread atrophy of a hemisphere follows on any large cerebral lesion, not on softening in particular; it follows clot, for example, and it may follow tumour, as is evident in some very long-standing cases. I say in some very long-standing cases, for, of course, in most cases of tumour, as we see them post mortem, from the added bulk and from œdema of the brain about the tumour, the convolutions are flattened and the brain looks bigger. Probably here there is concealed atrophy. In cases of autopsy in adults who have been hemiplegic from infancy, we may find, besides the local paralyzing lesion, extreme wasting of one cerebral hemisphere. This is, I think, a consequence of the local damage which caused the hemiplegia. There was at first, I suppose,

[1] The Lancet, April 29, 1871.

softening by thrombosis of a branch or of the trunk of the middle cerebral artery. This local softening is afterwards represented by a cyst. The atrophy is consecutive and secondary.

The mental symptoms following late (weeks or months) after an attack of hemiplegia are more slowly developed than those following immediately, hence they are very different. The deterioration shows itself both intellectually and emotionally. There begins to be loss of power of connected thought on difficult, novel, and complex subjects, or, speaking simply, the patient is soon confused; there are also peevishness and selfishness. Both these are highly characteristic of slow mental degradation. This is just what one would expect *a priori*. From a process so uniform as atrophy of the brain you would expect failure to begin slowly, and, on the Principle of Dissolution, that it would affect first the highest and most special of all faculties, or we may say those faculties last acquired both by the race and the individual. These faculties are power of abstract reasoning (intellectual) and sentiment of justice (emotional).[1] That these two faculties are the most special is seen by the fact that young children and savages have little or none of either. It is said that Australian savages have not even such abstract words as tree, red, justice, etc. Now, inferior people among the civilized are inferior because these faculties are little developed in them. I repeat, these are the two faculties developed latest; they fail first in cerebral disease where the whole brain is slowly implicated. So far for the principle of Dissolution, under which comes the negative condition. Next for the positive symptoms. The process of dissolution which directly causes the negative indirectly causes, or rather permits, the positive.

Patients with general cerebral deterioration, like persons naturally inferior, are easily excited; for by saying that they have defect in their highest processes, we say that they have defect in their controlling or inhibiting processes. To be easily excited is to have little controlling or inhibitory power. Here again comes in the Principle of loss of Control. Every healthy man has all animal passions and instincts, but the properly organized man has them under control of well-developed higher faculties. Thus chastity is not the absence of sexual feeling, but the having it deep under the control of higher feelings. Here you see that the principle of the same explanation holds good as was given of the mental symptoms following soon after local softening. In the production of the class of symptoms just considered, the process of dissolution is very slow, and thus the control is slowly removed. The suddenness and rapidity with which control is removed is of vast importance; the more rapidly it is removed the more activity is there of the centres uncontrolled. Thus, in the epileptic maniac (the most furious of all maniacs) the control has been removed very suddenly and with great rapidity.

Defective sense of justice seems too fine a phrase for selfishness, peevishness, and greediness; but it is really a correct statement of them. The feeling of justice is regard for others. Of course the results of disease are as different as are the persons to whose brains disease comes. For example, inferior men who have little sense of justice while in health, are made very irritable by trifles and easily take offence. They would soon, even by a non-cerebral illness, display bad temper, greediness, etc. Then some men have a feeling of justice in excess: generosity is excess of justice; generosity is not simply an easy, soft, careless nature. Such men are less likely to become selfish and greedy when the brain begins to fail. They have a reserve of good faculty. Defective power of abstract reasoning also seems too fine a phrase for the confused mental state of many of our hemiplegic patients. But it is really a correct expression; I don't mean that they have difficulty in reasoning on abstruse subjects. What puzzles them is connected thought about things out of their routine, and which are not simple and actually before them. Persons of congenitally inferior minds are easily confused by anything complex, especially if it be novel.

[1] See Herbert Spencer's Study of Sociology, chap. xv.

So, then, when general mental symptoms follow weeks or months after local softening, a sufficient explanation is given, not by the softening, which may not increase in extent as the symptoms develop, but by the slow consecutive atrophy.

To resume. Excluding softening about tumours and softening from gross causes similarly obvious, I know nothing of softening of the brain except that resulting from blocking of cerebral vessels. This, you will see, is saying, in effect, that softening of the brain is local; there is a softening of some part because a vessel supplying that part is blocked up. I have used in a former part of this lecture the expression "extension of softening." I wish to remark that, excluding softening about tumours, softening from injuries, etc., I know nothing of "extension of softening" except in the very simple sense of its resulting from new blockings up of arteries near those formerly blocked up.

The softening I have to speak of is a partial necrosis of brain mostly in arterial districts. Hence the first and essential thing in the study of softening is the study of the process and results of blocking up of arteries. It is exceedingly important to realize that it *is* a question of *arteries*. See how it bears on hereditary transmission of disease. Hemiplegia from local softening occurring in a person whose family has been subject to nervous disorders is no evidence whatever to show an inheritance of nervous disease, for the simplest possible reason that hemiplegia so caused is not strictly of nervous origin. The same remark applies to hemiplegia from clot, and thus it applies to nearly all cases of hemiplegia, for this form of palsy arises in the vast majority of instances from bad cerebral arteries; they break or get blocked up. Hemiplegia is the common arterio-cerebral symptom partly because an embolus can more easily get into the middle cerebral artery for anatomical causes, and partly because this vessel lies more directly in the way of strain from the heart, and thus is the most diseased and the most easily ruptured. To show how very mechanical, so to speak, the reason is for the predominance of this arterio-cerebral symptom, I mention that Prevost and Cotard found that tobacco-seeds injected into a dog's carotid most often lodged in the animal's middle cerebral artery. In fact, I know of no evidence to prove that any form of hemiplegia is ever of direct nervous origin —*i. e.*, that the pathological changes causing it *begin in nervous tissue*. Hence it is, I think, very inaccurate to speak of a patient who suffers epilepsy as having a *tendency* to nervous diseases because his father or mother had such symptoms as hemiplegia, loss of speech, softening of the brain, etc. Your notions on hereditary transmission of nervous diseases will be confused unless you bear in mind that nearly all "nervous diseases" are really instances in which the pathological changes begin in *non-nervous tissue*. Very many of them begin in the compound tissue (artery), and many of them in the simple tissue (connective). Besides symptoms of arterial origin there are nervous symptoms from intra-cranial tumours (including syphilitic growths), abscesses, hydatid cysts, etc. All these are pathologically extra-nervous. So, in inquiring for evidence as to hereditary tendencies in a patient who had any kind of nervous affection, you try to get evidence as to general states; for what you really want most to know is to what tissue-changes the patient's family is prone. You only inquire about nervous symptoms in other members of the patient's family (paralysis, etc.) as indirect evidence of this. You inquire, too, for rheumatism, gout, for facts as to renal disease, as well as ask if there be paralysis, insanity, etc. What you most wish to know is, what tendency there is to arterial (including cardiac) changes. My own belief is, that in this direction of inquiry we shall find out the pathology of the neuroses. I do not believe that chorea and epilepsy are nervous diseases in the sense that the pathological changes *begin in* nervous tissue.

In all the cases I have mentioned so far nerve-tissue *suffers*. It is not primarily at fault. As to the neuroses, remark that according to most physicians their pathology is unknown. We cannot say, then, that they are hereditary as *nervous affections* simply because they pervade a

family. Hemiplegia, apoplexy from cerebral hemorrhage, softening, etc., may pervade a family, but this is not evidence that that family is predisposed to nervous disease, for the simple reason that all the three so-called *nervous* diseases mentioned are affairs of arteries, not a whit more significant than defect of sight from retinal hemorrhage or epistaxis—arterial affairs too. In these cases it is more important to seek evidence of a tendency to arterial change than to note the occurrence of nervous disease or symptoms in the patient's blood relations.

Mental symptoms preceding cerebral softening.—It is important for another reason to bear in mind that cerebral softening is a question of arteries; for, since local softening is due to blocking up of arteries, it is hard to see how such so-called causes of softening as anxiety and overwork can be said to produce it. Such causes may help to produce the clinical grouping of symptoms, often called softening, of which I spoke at the beginning of my lecture. Fright and anxiety —which latter is only fright spread out thin—are, indeed, potent causes, especially in predisposed persons, of severe, very distressing, and long-continued nervous symptoms, such as sluggishness of mind and body, failure of power of attention, incapacity for sustained exertion, etc.; but I do not see how they can produce that *local* pathological change which is properly called softening. I do not deny that hemiplegia from softening (thrombosis) is often said by patients and their friends to have been preceded by anxiety or overwork—"caused by" such things, they will affirm. I think the most reasonable explanation is that the brain has been slowly getting into a condition in which it is easily excited and easily overworked; for in many such cases (softening from thrombosis) there is widespread degeneration of the cerebral arteries, and thus a worse nutrition throughout the brain, before the actual softening of some part results because one of these bad arteries has become blocked up. In some cases before the hemiplegia, the patient had manifestly become, as he would say, although not in these words, incapable of connected thought on any unusual matter, forgetful of things slightly out of his routine duties, and we should learn from his friends that he had become irritable and selfish. What I told you on failure of the two highest faculties in consecutive atrophy of the brain applies here. As a rule, the failure of mental power before softening is much slighter, and occurs more slowly; it is often spoken of by the patient's friends as indifference to business, fidgetiness, and "alteration" of disposition. These symptoms are no doubt, in persons who have degenerated arteries, the result of a uniformly bad state of the vessels of the brain. This prior condition of ill nutrition does not, so far as I know, lead to *general* softening, however slight we may suppose that softening to be.

You will now see that the causes of softening of the brain, properly considered, lead you to consider the state of the arterial system—lead you beyond the nervous elements of the nervous system. I repeat that very few nervous diseases are directly of nervous origin. A great part of our knowledge of the pathology of cerebral softening is in the answer to the question, Why do arteries become diseased, and thus "blockable"? Obviously if the softening of the brain be very limited in extent, and the symptoms therefore slight and transitory, the consideration of the patient's general bodily state is the really urgent matter. It would be a very poor thing to dwell with exaggeration on the hemiplegia, and ignore the state of the patient's heart, arteries, etc. It would be a very unclinical mind which could feel comfortable about a patient who was very "degenerate" simply because he had speedily got rid of hemiplegia. Inquire into degenerations of all systems of tissues, and examine all important organs. Nor must you suppose that you are taking a broad view of a case if you dwell exclusively on important superficial matters—state of digestion, sleep, and occupation. You will have your patient's sympathies in making such investigation, for he can, or he supposes he can, follow your inquiries and understand their bearing. It is best to begin

with the paralytic symptoms; then to consider the superficial, but very important, conditions; and, lastly, the deeper lying and most important evidence as to general pathological and pathogenetical states. The patient will wonder what you are at if, first of all, you examine his heart, urine, and arteries, when he comes to you because he cannot use his right arm and leg. Again, do not be illogical when the paralytic symptoms are slight and transitory. Suddenly occurring and transitory slight symptoms, such as affections of speech, unilateral numbness, may be owing to little foci of softening (or rather to thrombosis of small arteries, which in due time lead to local softening), just as much as permanent paralysis may be owing to extensive softening. Of course the slighter the symptoms the more need is there practically to consider many possibilities, for slight symptoms are not necessarily so easy of explanation as grave symptoms often are. After carefully considering all the causes I can think of, I feel warranted in attributing in many cases the slight and transitory nervous symptoms I have mentioned to local softening; for vessels of very small size may be blocked up as well as vessels of great size, and in correspondence there are foci of softening from the size of a pea upwards. Simple as this remark sounds, the principle deducible from it is not always borne in mind. Slight and transitory hemiplegia, which we are warranted in putting down to blocking up of very small arteries, is often hastily ascribed to general states of ill-health—for example, to affection of the liver, etc. In so explaining them you would have your patient's sympathies. He can, or he thinks he can, frame some sort of conception of "local congestion," "disorder of the liver," "suppressed gout," "overwork," "anxiety," etc., as causing his slight localized nervous symptoms. At any rate these explanations or these phrases are familiar to him. I think that the existence of any of these states gives no explanation of local and suddenly occurring nervous symptoms. These "explanations" are superficial, not simple. If a man have atheromatous arteries (blockable arteries) of all sizes, why should not slight and transitory symptoms be owing to blocking of very small arteries? Observe, the question of first importance in diagnosis is not as to the slightness or transitoriness of a symptom, but as to its localness; a local symptom must imply a local lesion of some sort. One reason for the frequent denial that local lesions exist in cases of *transitory* paralysis is that it is not widely known that there is Compensation in nervous organs, so that paralysis will pass off when the damage causing it is not altogether repaired. Thus, we exclude such so-called general causes as I have mentioned, and go on to consider special causes. At the same time it is only an inference that such slight and transitory paralytic symptoms depend on small foci of softening; for there is no, or practically no, morbid anatomy of such slight symptoms.—*Lancet*, Sept. 4, 1875.

MEDICAL NEWS.

ORIGINAL ARTICLE.

Pruritus Formicans.—Dr. William T. Thackeray of Yankton, Dakota, writes us as follows:—

In the January number of the *American Journal of the Medical Sciences*, I notice an article on Pruritus Formicans accompanying Pregnancy, by Horace V. Evans, M.D.

My attention was directed to this subject early in my professional life, and my treatment (upon the recommendation of the late Prof. S. Henry Dickson) has consisted simply of topical applications of cider vinegar; this plan has proved uniformly successful in all the cases that have come under my care.

DOMESTIC INTELLIGENCE.

International Medical Congress, Philadelphia, 1876.—The preliminary arrangements for the organization of the International Medical Congress, which is to convene in Philadelphia on the 4th day of September, 1876, are rapidly taking form, and the Centennial Medical Commission, which has charge of these duties, expects in a few days to issue a circular

giving the details of the plan of the Congress as far as already determined upon. We are informed that the prospects of the success of the Congress as a scientific body are already flattering. A large number of the leading medical minds of Europe are confidently expected to participate in its deliberations.

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American Public Health Association. Annual Meeting, 1875.—This important and useful Association will convene in Baltimore, Tuesday, November 9, 1875, in accordance with the adjournment of the previous annual meeting.

We have been favoured with the order of business adopted by the Executive Committee, but too late for insertion. From the list of papers to be read, it is evident that the meeting will be an instructive one.

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The Code of Ethics of the American Medical Association has been unanimously adopted by the Medical Society of Munich, and translated for the use of its members. —*Medical Record*, Oct. 9, 1875.

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State Board of Health of Georgia.—The State of Georgia, by an Act approved 25th February, 1875, established "a State Board of Health, for the protection of life and health and to prevent the spread of diseases in the State, and for other purposes." We have been favoured with a copy of the constitution and rules of this Board, of the address issued by it, and of the proceedings of its first meeting; also of the forms issued for returns to be made of births, marriages, deaths, vital statistics, etc.

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Harlem Lodge.—This is the title of an establishment recently purchased and improved by the *Trustees of the Maryland Inebriate Asylum*, and is situated near the city of Baltimore. It is designed to provide especially for a class of men who wish to recover and who will accept of the appliances which the Lodge furnishes. The rooms are private, and two physicians reside in the building. Special arrangements may be made for a few female opium patients. Applications to be made to Joseph Parrish, M.D., Baltimore P. O., Maryland.

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Starling Medical College, Columbus, Ohio. —Dr. JOHN H. POOLEY, of Yonkers, New York, has been appointed Prof. of Surgery in this school.

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Frederick D. Lente, M.D.—This experienced and skilful practitioner, in consequence of impaired health, has removed from Cold Spring, New York, and intends to reside during the winter months at Palatka, Florida, and during the summer months at Saratoga Springs, New York, (Grand Union Hotel) where he may be consulted professionally.

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Dr. Edward Warren.—We regret to learn that Dr. Edward Warren, originally of North Carolina, but subsequently of Baltimore, who, it will be remembered, some three years since accepted a position on the medical staff of the Army of the Khedive of Egypt, was attacked with malignant ophthalmia shortly after his promotion to the office of Surgeon-General of the Egyptian Army. The obstinacy of the affection compelled him to seek relief in Paris. At the end of six months of treatment he is left with one eye permanently enfeebled, and the probability of losing the sight if he return to Egypt. He has accordingly obtained permission to practise in Paris.

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The Physician's Visiting List for 1876.—Messrs. Lindsay & Blakiston have just issued this useful annual, the convenience of which to the practising physician is too well known to require more special notice.

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Physician's Office Case-Record and Prescription Blank Book.—This very convenient and useful book for recording cases and prescriptions is issued by the Case Record Company, Cincinnati. The price of the charts is very moderate (three dollars per hundred). They may be obtained from Dr. Geo. E. Walton, 224 Laurel St., Cincinnati.

OBITUARY RECORD—Died, at Sing Sing, N. Y., Sept. 23d, of peritonitis following typhoid fever, ERNEST KRAKOWIZER, M.D., aged 54. Dr. K. was born in Upper Austria, emigrated to this country in 1850, and finally settled in the city of New York, where he acquired a high reputation for his surgical knowledge and skill, obtained a large practice, and became greatly esteemed. He was one of the surgeons of the New York Hospital and of the Mount Sinai Hospital, and had many distinguished professional offices conferred on him.

FOREIGN INTELLIGENCE.

Carbazotate of Ammonia as a Substitute for Quinia.—Dr. I. SLANE, of Goalpera, states that his supply of quinia being nearly exhausted, and the indigenous antiperiodics failing, he was compelled to cast about him for a remedy with which to supplement his small stock of quinia. He determined to give the carbazotate of ammonia a trial, and proceeded to administer it to his fever cases. Up to the time of his writing (three months' experience of it) he had obtained very gratifying results. He finds whilst it is far cheaper than quinia, the dose is much smaller. The largest quantity he gave in *any* case was 8 grains in the twelve hours. The salt has no perceptible action in increasing the appetite, but singularly enough, like quinia, after a few doses it produces tinnitus aurium and slight deafness. The urine becomes tinged of a deep orange colour, which stains the clothes under its use, and the skin and conjunctiva may acquire a jaundiced hue, as occurred in two of his cases, but the discoloration soon passed off. He gave it in pill with extract of gentian; but it may also be given in solution, as it is perfectly soluble in water; and the liquor strychniæ may then be combined with it. Its intensely bitter taste is, however, an objection to its use in solution. Combined with a quarter of a grain of extract of nux vomica, its efficacy seemed to be much increased.—*Practitioner*, September, 1875, from *Indian Medical Gazette*, April 1, 1875.

Therapeutic Properties of Salicylic Acid.—Some very important observations by M. WAGNER on salicylic acid appear in a recent number of the *Journal für Practische Chemie*. His results are thus summarized by Dr. Stevenson in the August number of the *Journal* of the Chemical Society: (1) Salicylic acid is superior to phenol (carbolic acid) as a disinfectant for fresh wounds and old sores. (2) A disinfecting action is insufficient for venereal sores, and corrosion is requisite. (3) In eczema of the head and face, with discharge, salicylic acid is extraordinarily efficacious, presumably because it quickly destroys the contagium. (4) In all cases where fermentative changes occur in the contents of the alimentary canal, salicylic acid acts more efficaciously than other antiseptic substances, since it can be administered in larger doses. (5) Its use is highly promising as a prophylactic in all diseases in which it is believed that the morbid processes are connected with microscopic organisms. In diphtheria, not only is salicylic acid a powerful restorative remedy, but it also appears to shorten the course of the disease.—*Lancet*, Sept. 18, 1875.

Antifebrile Action of Salicylic Acid.—Herr SENATOR read, before the same Society, a paper on the above subject, giving an account of numerous trials which he had made in consequence of the recommendation published by Fürbringer and Buss (*Centralblatt*, 1875, No. 18), of the employment of salicylic acid as a means of abating temperature in fevers. He had especially tried it in the hectic of phthisis, having made fifty observations in ten cases. He found that under its use the temperature was undoubtedly diminished, but not so certainly as under the influence of quinia. In some of the cases its influence was quite surprising, while in others it was doubtful or not recognizable. He thinks that these negative results may be due to his having begun with too small doses, or to the mode of administration. The effect was much more marked in intermittent fever. In five out of ten cases of this, the cure was complete after once or twice employing

the medicine. The other five, being dispensary patients, did not reappear. The dose was from one to two grammes, either given altogether or in separate doses shortly before the paroxysm; correspondingly smaller doses being given to children. At first it was given as a powder, but later in warm water and glycerine, viz.—two parts of acid to 200 of boiling water, and sufficient glycerine to effect the solution. The mixture was warmed each time before it was taken. A good deal of discussion ensued upon the mode of administration, most speakers considering the forms of powder or emulsion undesirable, while the quantity of water required, on account of the great insolubility of the acid, was also objectionable. Glycerine was generally recommended, and it was stated that fifty parts of this and fifty of water constituted a vehicle that held the acid in permanent solution.—*Ibid.*

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Treatment of Hydatid Disease of the Liver.—The prevalent belief that simple tapping of a hydatid cyst in the liver is sufficient for its radical cure, owing to the death of the echinococcus following the escape of its fluid contents, is not entertained by M. VERNEUIL, the eminent surgeon attached to the Hôpital de la Pitié (*Revue de Thérapeutique Medico-Chirurgicale*, August 15th). He believes that the remains of the cyst acting as a foreign body, if they do not suppurate themselves, yet set up suppuration in their vicinity. Recognizing also the danger of peritonitis from escape of the hydatid fluid into the abdominal cavity, he does not adopt the so-called method of Recamier—viz., external application of caustic—nor other means of promoting adhesion; but he begins by puncturing the most prominent part of the cyst with a large trocar and canula. He then introduces through the canula a large gum-elastic catheter. Adhesions take place between the visceral and parietal peritoneum, and, there being constant means of escape, there is no retention of the fluid. Repeated injections of disinfectants, as solutions of carbolic acid or alcohol, are made into the cyst, the walls of which are thus allowed to slowly contract.—*Lancet*, Sept. 18, 1875.

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Subcutaneous Injection of Carbolic Acid in Rheumatism.—At the Berlin Medical Society, Herr SENATOR read a paper which will hereafter be published at length, entitled "The Local Employment of Subcutaneous Carbolic Acid Injections in Rheumatic Polyarthritis," containing an account of the results of the practice which, on the recommendation of Kunze, he has followed in some cases in private practice, and in many in the Augusta Hospital. At first he used a solution of 1 per cent., and later of 2 or 3 per cent., injecting this in the vicinity of the various joints affected. These injections had never been attended by any ill consequences, and had often given rise to the disappearance of the pain and other local symptoms. Complications and relapses of the disease were, however, not prevented. Herr Goldbaum stated that he had for several years been employing subcutaneous injections in articular rheumatism, as also in muscular rheumatism, and the anæsthetic effect has been remarkable, continuing for five or six hours. As the patient complains chiefly of the excessive pain and the immovability of the joint, this means is much to be recommended, for by it not only is the pain relieved, but the joint becomes movable, such mobility remaining as long as the pain is absent. He believes that in several cases a favourable influence is exercised on the general course of the disease. The efficacy of the practice is especially seen in muscular rheumatism, so that patients who were motionless from lumbago were after the injections able to move; and after these had been repeated several times the affection disappeared. In answer to a question from Herr v. Langenbeck as to whether the general febrile action was influenced, Herr Senator said that he could not pronounce on this point, as in articular rheumatism the febrile action depended on the affections of the joints, rising or falling with these. The carbolic acid, he further observed, is only a palliative. He has used the 3 per cent. solution with

great advantage in a case of ischias, but he has met with no success in chronic articular rheumatism. Herr Goldbaum observed that in no form of neuralgia in which he has tried the remedy has any benefit been derived, so that when no result attends the use of the remedy, the conclusion he comes to is, that a nervous, not a muscular affection, is under treatment. He has, however, found that some of a 2 per cent. solution put into the ear effectually relieves otitis externa, the patient renewing the application as soon as the pain returns. Herr Citron, while confirming what had been said of the anæsthetic effect of the acid in rheumatism, said that on taking the temperatures he had found no effect produced on these. Herr Waldenburg called attention to an old and very simple palliative that had very seldom failed him, viz., pouring ether over the part. Applied as ether-spray he had found it less efficacious. Herr Retzlag, having suffered himself from the disease, had found the ether of no service, except during the moment of application. He had derived most benefit from blisters kept on for twelve hours, until vesicles had formed, which were then opened. Herr Frankel had not tried carbolic acid, but among other local remedies he preferred the immobilization of the joint for ten or twelve hours, when the pain ceased.—*Med. Times and Gaz.*, Sept. 18, from *Berlin. Med. Woch.*, Aug. 16, 1875.

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Bromoform as an Anæsthetic.—Bromoform in a pure state, as obtained by Rabuteau from the action of an alkali upon bromal, appears in the form of a clear, very heavy, and agreeably smelling fluid. Iodine dissolves in it, and colours it of a carmine-red colour. The mode of action of bromoform resembles that of chloroform very closely. When administered in the form of vapour, there is a period of excitement which is followed by deep sleep, in which there is complete anæsthetic relaxation of all the muscles and dilatation of the pupil. The awakening from the effects of the agent takes place tolerably rapidly, even if the inhalation has been continued for half an hour. Rabuteau hopes that it will come into use.—*Practitioner*, September, 1875, from *Société de Biologie*, Jan. 1875.

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New Acid found in the Urine after Ingestion of Chloral Hydrate.—Musculus and De Mering have recently published, in the *Comptes Rendus*, an account of a curious acid which seems to be formed under the above conditions. Examining the urine of persons who were taking sixty to eighty grains of chloral hydrate per diem, they found it strongly acid, reducing copper solutions and rotating polarized light to the left. The acid is thrown down by basic lead acetate, and forms stellate crystals like those of tyrosine. It is a powerful acid, decomposing carbonates with effervescence, while its salts are unaffected by acetic acid. It is very soluble in water and alcohol, but not in ether, and reduces salts of copper, silver, and bismuth, and decolourizes sulphate of indigo. It is said to contain 31.60 C, 4.36 H, and 26.7 Cl. All its salts are soluble in water except the basic lead salt, but insoluble in absolute alcohol. The discoverers propose to call it provisionally *urochloralic acid*. It would seem from this discovery that chloral, like benzoic acid, undergoes material change in the organism, and is excreted in a different form, though some passes unaltered. The quantity of the new acid found was from ten to twelve grammes per litre of urine.—*Lancet*, Sept. 18, 1875.

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International Medical Congress at Brussels.—The meeting of this Congress was eminently brilliant and successful. It was opened on Sunday, Sept. 19th, in the great Hall of the Ducal Palace, by M. Vleminex of Brussels, Provisional President; His Majesty the King of the Belgians gracing the ceremony by his presence. On Monday a reception was given to the members by the communal authorities at the Hôtel de Ville; on Thursday there was a brilliant *soiree* at the official residence of the Minister of the Interior, and the Congress closed on Saturday by a farewell banquet at the Hôtel de Ville.

The following officers were elected: President, M. Vleminex, of Brussels.

Honorary Presidents, Langenbeck, of Germany; Critchett and Bowman, of England: Hebra and Sigmund, of Austria; Bouillaud, Verneuil, Larrey, and Jaccoud, of France; Semmola and Palasciano, of Italy; Aschman, of Luxembourg; Marcowitz, of Roumania; Ahmet, of Turkey; Donders, of Holland; Manayra and Pasquali, of Italy; Nicolayew, of Russia; Van Cappelle and Eyeling, of Holland; Schnitzler, of Austria; Gross, of Hungary; G. Bergmann, of Sweden; Harwood, of New York, and Adrian, of Indiana, U. S.; Madjen, of Copenhagen; and Petersen, of Denmark.

Vice-Presidents, Deroubaix and Crocq.

Secretary-General, M. Warlomont.

Recording Secretaries, MM. Duwez and Verriest.

The work of the Congress was distributed among nine sections. Each section met in the morning at 10 o'clock to discuss certain scientific questions, the present status of which were set forth by reporters designated in advance. The provisional conclusions contained in these reports were scrutinized seriatim, modified when deemed desirable, and then reported by the Sections to the Congress in general meeting, and each conclusion there voted on separately.

The general meetings of the Congress were devoted—1st, to verbal communications and to reports of sections, and, in case of disagreement in the latter, to their discussion; 2d, to conferences and to communications upon questions of general medical interest which were not upon the programme.

The various sections did a goodly amount of scientific work, which, in abstract, will be laid before our readers hereafter. The Congress adjourned on the 25th of September to meet in Switzerland in 1877. Before adjournment, however, it voted to send delegates to the International Medical Congress to be held in Philadelphia in September, 1876.

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School of Anthropology and Medical Geography.—"Thanks to the active initiative of Prof. Paul Broca, and the ready concurrence of the Faculty of Medicine, an 'École d'Anthropologie' has just been founded at Paris. The École Pratique is to be employed for its purposes, and it will be supported solely by private subscriptions. The subjects of the various courses of lectures have been settled, and the following is the list of the professors: Anatomical Anthropology, M. Broca; Ethnological Anthropology, M. Dally; Linguistical Anthropology, M. Hovelacque; Prehistorical Anthropology, M. De Mortillet; Biological or General Anthropology, M. Topinard. Moreover, a very distinguished *confrère*, to whom we owe a remarkable work 'On the Influence of Atmospheric Pressure on the Life of Man' (M. Paul Bert), has placed at the disposal of the new school a sum of 40,000 francs to meet the expenses of a supplementary course on Demography and Medical Geography, and for this chair M. Bertillon has been chosen."—*Gaz. Hebdomadaire*, Sept. 24, 1875.

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Obituary Record.—Died, at Norwich, September 25, from the after-effects of lithotomy on a system weakened by constitutional disease, aged sixty-three, John Hughes Bennett, M.D., late Professor of the Institutes of Medicine in the University of Edinburgh. Prof. B. was a man of mark in the profession and an author of a number of professional works. His latest contribution to our science was his valuable report on the Antagonism of Medicines.

—— at Boulogne, September 18, 1875, Dr. Duchenne, in the seventieth year of his age. He is well known by his two great works, "*Traité de electrisation localisée*," and "*La Physiologie des mouvements*."

—— at Pavia, September 10, aged 75, Luiga Porta, Professor in the University of Pavia, one of the most illustrious of Italian surgeons, and the author of a most important work on the diseases of the bloodvessels.

—— at Hanover, September 8, Prof. J. C. Jüngken, aged 82. Prof. J. was the father of the Berlin Medical Faculty, and had great renown as an operating surgeon. It is to him that was chiefly owing, in Germany, the scientific culture of ophthalmology.

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